U.S. Department of Labor

Office of Administrative Law Judges
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Issue Date: 10 December 2002

In the Matter of

MRS. ROSALIE DOLORES OWENS,
on behalf, and as widow, of
MR. ANDREW C. OWENS
Claimant

Case No.: 2002 BLA 111
2002 BLA 112

v.

SOUTH HOLLOW COAL CO./ROCKWOOD INS.
Employer/Carrier
and
J & J COAL COMPANY/OLD REPUBLIC INS.
Employer/Carrier
and
BIG BLUE COAL COMPANY/OLD REPUBLIC INS.
Employer/Carrier
and
DIRECTOR, OFFICE OF WORKERS' COMPENSATION PROGRAMS
Party in Interest

Appearances: Mrs. Rosalie D. Owens
Pro Se

Mr. Russell Vern Presley, II, Attorney
For the Employer/Carrier

Before: Richard T. Stansell-Gamm
Administrative Law Judge

DECISION AND ORDER - DENIAL OF BENEFITS

This matter involves a claim filed by Mr. Andrew C. Owens prior to his death and a survivor claim filed by Mrs. Rosalie D. Owens for benefits under the Black Lung Benefits Act, Title 30, United States Code, Sections 901 to 945 ("Act"). Benefits are awarded to persons who are totally disabled within the meaning of the Act due to pneumoconiosis, and to survivors of persons who died due to pneumoconiosis. Pneumoconiosis is a dust disease of the lung arising from coal mine employment and is commonly known as "black lung" disease.

Pursuant to a Notice of Hearing, dated March 26, 2002, I set a hearing date of June 25, 2002 for this case in Abingdon, Virginia (ALJ I).[1] On June 11, 2002, Mrs. Owens waived her right to a hearing and requested a decision on the record. In light of Mrs. Owens' request, and in the absence of an objection by counsel for the Employer, I cancelled the scheduled hearing and provided the parties an opportunity to present additional exhibits subject to objection (ALJ II). Subsequently, counsel for the Employer submitted four exhibits, which I have marked EX 1 to EX 4. Since the Claimant has entered no evidentiary objection, I now admit EX 1 to EX 4 into evidence. Consequently, my decision in this case is based on all documents in the record: DX 1 to DX 84, and EX 1 to EX 4.

ISSUES

1. Whether Mr. Owens suffered from pneumoconiosis.

2. If Mr. Owens had coal workers' pneumoconiosis, whether his death was due to pneumoconiosis.

3. Whether Mr. Owens established a material change in conditions since the denial of his second claim for benefits on August 13, 1997.

4. If Mr. Owens established a material change in conditions, whether he was entitled to disability benefits under the Act.

Coal Miner's Background

Born March 20, 1929, Mr. Andrew C. Owens married Mrs. Rosalie Dolores Hay on May 30, 1947 (DX 6). Mr. Owens started mining coal in 1947 (DX 2). For approximately 30 years, he worked as an underground coal miner running a motor, loading coal, and eventually serving as a foreman before he quit work in 1984 due to health problems (DX 10). Sadly, Mr. Owens passed away on September 17, 2000 (DX 58).

Procedural Background

Mr. Owens' Claims

First Claim

Mr. Owens filed his first claim for benefits under the Act on June 11, 1973 with the Social Security Administration (DX 46-1). In April 1978, Mr. Owens requested that his claim be reviewed by the U.S. Department of Labor ("DOL") (DX 46-25). Eventually, DOL denied his claim on May

[1]The following notations appear in this decision to identify exhibits: DX - Director exhibit; EX - Employer exhibit; and ALJ - Administrative Law Judge exhibit.

13, 1981 for failure to establish pneumoconiosis and total disability due to black lung disease (DX 46-29).

Second Claim

Mr. Owens filed his second (duplicate) claim on April 18, 1997 (DX 47-1). On August 13, 1997, DOL informed Mr. Owens that his claim was denied because the evidence did not establish: pneumoconiosis, total disability due to pneumoconiosis, and a material change in condition (DX 47-21).

Third, and Present, Claim

On May 4, 1999, Mr. Owens filed with DOL another duplicate claim, representing his third attempt to obtain benefits under the Act (DX 1). On September 20, 1999, based on a pulmonary examination, the District Director rendered an initial decision finding Mr. Owens totally disabled due to coal workers' pneumoconiosis and thus entitled to benefits under the Act (DX 26). After the named coal companies contested his entitlement (DX 27 and DX 28), the District Director considered additional information but remained convinced that Mr. Owens was entitled to black lung disability benefits. Consequently, he informed the parties on January 18, 2000 of his decision (DX 39 and DX 40). On January 28, 2000, the responsible operators contested the award of benefits and requested a formal hearing before the Office of Administrative Law Judges ("OALJ") (DX 41). As a result, the District Director initiated interim payments and forwarded the case to the OALJ on March 22, 2000 (DX 45 and DX 49).

Initially, a hearing was scheduled for August 2000 (DX 51) but it was continued when an attorney entered an appearance on Mr. Owens' behalf and requested a continuance (DX 56). The hearing was rescheduled for January 2001. Unfortunately, Mr. Owens passed away on September 17, 2000 (DX 58). When Mr. Owens' attorney submitted evidence concerning Mr. Owens' passing, counsel for the Employer requested that the rescheduled hearing be continued for development of additional evidence and consideration of any survivor claim (DX 59). In response, Administrative Law Judge Edward T. Miller issued an Order of Remand dated December 26, 2000 to allow for the development of additional evidence and consideration of a potential survivor's claim because of the miner's death (DX 61). Eventually, in May 2001, based on the evidence developed following the remand of the claim, the District Director denied Mr. Owens' duplicate claim for failure to establish black lung disease, total disability due to black lung disease and a material change in condition (DX 65).

Mrs. Owens' Survivor Claim

Mrs. Rosalie Owens filed her own application for benefits on January 1, 2001 (DX 66). However, also in May 2001, the District Director denied Mrs. Owens' survivor claim because the evidence failed to establish either the presence of pneumoconiosis or that the disease caused the miner's death (DX 76).

On June 2, 2001, Mrs. Owens contested the denials of her claim and Mr. Owens' claim for disability benefits and requested a formal hearing before OALJ (DX 79 and DX 81).[2] On October 22, 2001, the District Director forwarded both claims to the OALJ for a hearing (DX 84). As noted above, I have granted Mrs. Owens' request for a decision on the record.

FINDINGS OF FACT AND CONCLUSIONS OF LAW

The claims of Mr. and Mrs. Owens have a crucial issue in common - whether Mr. Owens had coal workers' pneumoconiosis. To facilitate my adjudication of both claims, I will first consider Mrs. Owens' survivor claim. A favorable finding on the issue of pneumoconiosis in her case would then also establish the necessary change in condition sufficient to satisfy the regulation requirement associated with the reconsideration of Mr. Owens' duplicate claim.

Mrs. Owens' Survivor Claim

Under the Act, and the implementing regulations, 20 C.F.R. § 718.205 (a), benefits are provided to eligible survivors of a miner whose death was due to pneumoconiosis. To obtain benefits, a surviving claimant must prove by a preponderance of the evidence several facts. First, the claimant must establish eligibility as a survivor. A surviving spouse may be considered eligible for benefits under the Act if she was married to, and living with, the coal miner at the time of his death, and has not remarried.[3]

Next, the claimant must prove the coal miner had pneumoconiosis.[4] "Pneumoconiosis" is defined as a chronic dust disease arising out of coal mine employment. The regulatory definitions include both clinical pneumoconiosis (the diseases recognized by the medical community as pneumoconiosis) and legal pneumoconiosis (defined by regulation as any chronic lung disease arising out of coal mine employment) 20 C.F.R. § 718.201 (a) (1) and (2). The regulation further indicates that a lung disease arising out of coal mine employment includes "any chronic pulmonary disease or respiratory or pulmonary impairment significantly related to, or substantially aggravated by, dust exposure in coal mine employment." 20 C.F.R. § 718 (b). As courts have noted, under the Act, the legal definition of pneumoconiosis is much broader than medical pneumoconiosis. *Kline v. Director, OWCP*, 877 F.2d 1175 (3d Cir. 1989).

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[2]Due a change in his location, Mr. Owens' counsel at the time withdrew from the case (DX 82).

[3]20 C.F.R. § 718.4 indicates that the definitions in 20 C.F.R. § 725.101 are applicable. 20 C.F.R. § 725.101, in turn, refers to the term "survivor" as used in Subpart B of Part 725. 20 C.F.R. § 725.214 then sets out the espousal relationship requirements and 20 C.F.R. § 725.215 describes the dependency rules. According to § 725.214 (a) the spousal relationship exists if the relationship is a valid marriage under state law. Under § 725.215(a), a spouse is deemed dependent if she was residing with the miner at the time of his death.

[4]20 C.F.R. § 718.205 (a) (1) and *see Trumbo v. Reading Anthracite Co.*, 17 B.L.R. 1-85 (1993).

Third, once a determination has been made that a miner has pneumoconiosis, it must be determined whether the coal miner's pneumoconiosis arose, at least in part, out of coal mine employment.[5] If a miner who is suffering from pneumoconiosis was employed for ten years or more in one or more coal mines, there is a rebuttable presumption that pneumoconiosis arose out of such employment.[6] Otherwise, the claimant must provide competent evidence to establish the relationship between pneumoconiosis and coal mine employment.[7]

Finally, the surviving spouse has to demonstrate the coal miner's death was due to pneumoconiosis.[8]

In summary, a survivor claim filed after January 1, 1982 must meet four primary elements for entitlement. The claimant bears the burden of establishing these elements by a preponderance of the evidence. If the claimant fails to prove any one of the requisite elements, the survivor claim for benefits must be denied. *Gee v. W. G. Moore and Sons*, 9 B.L.R. 1-4 (1986) and *Roberts v. Bethlehem Mines Corp.*, 8 B.L.R. 1-211 (1985). The four elements are: (1) the claimant is an eligible survivor of the deceased miner; (2) the coal miner suffered from pneumoconiosis; (3) the coal miner'spneumoconiosis arose out of coal mine employment; and, (4) the coal miner'sdeath was due to coal workers' pneumoconiosis.

Eligible Survivor

Based on the Owens' marriage certificate (DX 6) and Mr. Owens' death certificate showing his continuing marriage to Mrs. Owens (DX 58 and DX 68), I find Mrs. Rosalie Owens is an eligible survivor under the Act. Accordingly, Mrs. Owens has established the first requisite element of entitlement.

Issue No. 1 - Presence of Pneumoconiosis

The next entitlement element that Mrs. Owens must prove is that Mr. Owens had pneumoconiosis. According to 20 C.F.R. § 718.202, the existence of pneumoconiosis may be established by four methods: chest x-rays (§ 718.202 (a)(1)), autopsy or biopsy report (§ 718.202 (a)(2)), regulatory presumption (§718.202 (a)(3)),[9] and physician medical opinion (§718.202 (a)(4)).

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[5]20 C.F.R. §§ 718.203 (a) and 718.205 (a)(2).

[6]20 C.F.R. § 718.203 (b).

[7]20 C.F.R. § 718.203 (c).

[8]20 C.F.R. § 718.205 (a)(3).

[9]If any of the following presumptions are applicable, then under 20 C.F.R. § 718.202 (a)(3) a miner is presumed to have suffered from pneumoconiosis: 20 C.F.R. § 718.304 (if complicated pneumoconiosis is present then there is an irrebuttable presumption the miner is totally disabled due to pneumoconiosis); 20 C.F.R. § 718.305 (for claims filed before January 1, 1982, if the miner has fifteen years or more coal mine employment, there is a

(continued...)

Since the record does not contain evidence that Mr. Owens had complicated pneumoconiosis and Mrs. Owens filed her claim after January 1, 1982, a regulatory presumption of pneumoconiosis is not applicable. As a result, Mrs. Owens will have to rely on chest x-rays, biopsy/autopsy evidence, and medical opinion to establish the presence of pneumoconiosis. Additionally, under the court's guidance in *Island Creek Coal Co. v. Compton*, 211 F.3d 203 (4th Cir. 2000), I must consider the chest x-rays, the biopsy/autopsy evidence, and medical opinion together to determine whether Mr. Owens had pneumoconiosis.

Chest X-rays

Date of x-ray	**Exhibit**	**Physician**	**Interpretation**
7/10/1973	DX 46-20	Knieton	Negative for pneumoconiosis
(same)	DX 46-21	Dr. Morgan	Negative for pneumoconiosis
4/30/1979	DX 46-22	Dr. Wright	Negative for pneumoconiosis
5/23/1979	DX 46-15	Dr. Hernandez	Clear lungs
6/3/1979	DX 46-15	Dr. Westmoreland	Mild interstitial markings
6/4/1979	DX 46-15	Dr. Hernandez	Fine interstitial markings
6/12/1979	DX 46-15	Dr. Westmoreland	Minimal increase in markings
8/18/1979	DX 46-15	Dr. Hernandez	Pneumonic infiltrate, right side
8/21/1979	DX 46-15	Dr. Westmoreland	Faint infiltrate, right side
5/26/1981	DX 46-24	R.O.G., B[10]	Negative for pneumoconiosis, profusion 0/1,[11] type t opacities[12]

[9](...continued)
rebuttable presumption that total disability is due to pneumoconiosis); and 20 C.F.R. § 718.306 (a presumption when a survivor files a claim prior to June 30, 1982).

[10]B - B Reader; and BCR - Board Certified Radiologist. These designations indicate qualifications a person may possess to interpret x-ray film. A "B Reader" has demonstrated proficiency in assessing and classifying chest x-ray evidence for pneumoconiosis by successful completion of an examination. A "Board Certified Radiologist" has been certified, after four years of study and an examination, as proficient in interpreting x-ray films of all kinds including images of the lungs.

[11]The profusion (quantity) of the opacities (opaque spots) throughout the lungs is measured by four categories: 0 = small opacities are absent or so few they do not reach a category 1; 1 = small opacities definitely present but few in number; 2 = small opacities numerous but normal lung markings are still visible; and, 3 = small opacities very numerous and normal lung markings are usually partly or totally obscured. An interpretation of category 1, 2, or 3 means there are opacities in the lung which may be used as evidence of pneumoconiosis. If the interpretation is 0, then the assessment is not evidence of pneumoconiosis. A physician will usually list the

(continued...)

Date of x-ray	Exhibit	Physician	Interpretation
8/23/1996	DX 35	Scott, BCR, B	Negative for pneumoconiosis; few peripheral scars upper lungs compatible with healed TB (tuberculosis)
(same)	DX 35	Wheeler, BCR, B	Negative for pneumoconiosis; probable few tiny scars or calcified granulomata compatible with healed TB
5/6/1997	DX 47-11	Kanwal	Negative for pneumoconiosis
(same)	DX 47-12	Navani**,** BCR, B	Negative for pneumoconiosis
(same)	DX 47-13	Gaziano, B	Negative for pneumoconiosis
9/28/1997	DX 35	Scott, BCR, B	Negative for pneumoconiosis; few peripheral scars upper lungs compatible with healed TB
(same)	DX 35	Wheeler, BCR, B	Negative for pneumoconiosis; probable few tiny scars or calcified granulomata compatible with healed TB; hyperinflation lungs compatible with deep breath or emphysema.
9/4/1998	DX 35	Scott, BCR, B	Negative for pneumoconiosis; few tiny peripheral scars upper lungs compatible with healed TB
(same)	DX 35	Wheeler, BCR, B	Negative for pneumoconiosis; probable few tiny scars or calcified granulomata compatible with healed TB; hyperinflation lungs compatible with deep breath or emphysema.
2/11/1999	DX 62	Scott, BCR, B	Negative for pneumoconiosis

[11](...continued)
interpretation with two digits. The first digit is the final assessment; the second digit represents the category that the doctor also seriously considered. For example, a reading of 1 / 2 means the doctor's final determination is category 1 opacities but he considered placing the interpretation in category 2. Or, a reading of 0/0 means the doctor found no, or few, opacities and didn't see any marks that would cause him or her to seriously consider category 1. Additionally, according to 20 C.F.R. § 718.102 (b), a reading of 0/1 does not constitute evidence of pneumoconiosis.

[12]There are two general categories of small opacities defined by their shape: rounded and irregular. Within those categories the opacities are further defined by size. The round opacities are: type p (less than 1.5 millimeter (mm) in diameter), type q (1.5 to 3.0 mm), and type r (3.0 to 10.0 mm). The irregular opacities are: type s (less than 1.5 mm), type t (1.5 to 3.0 mm) and type u (3.0 to 10.0 mm). JOHN CRAFTON & ANDREW DOUGLAS, RESPIRATORY DISEASES 581 (3d ed. 1981).

Date of x-ray	Exhibit	Physician	Interpretation
(same)	DX 62	Wheeler, BCR, B	Negative for pneumoconiosis; probable few tiny scars from healed TB
5/27/1999	DX 13, DX 71	Paranthaman, B	Positive for pneumoconiosis; profusion 1/0, type q/t opacities; emphysema
(same)	DX 14, DX 15, DX 71	Gaziano, B	Positive for pneumoconiosis; profusion 1/0; type t/q/u opacities
(same)	DX 36	Scott, BCR, B	Negative for pneumoconiosis; possible few peripheral scars right upper lung compatible with healed TB
(same)	DX 36	Wheeler, BCR, B	Negative for pneumoconiosis; probable few tiny scars
9/27/1999	DX 35	Scott, BCR, B	Negative for pneumoconiosis; calcified granuloma left apex
(same)	DX 35	Wheeler, BCR, B	Negative for pneumoconiosis; probable 6 mm oval calcified granuloma left apex and few tiny scars in lateral; probable decreased left lower lung markings compatible with emphysema
12/8/1999	DX 37	Fino, B	Negative for pneumoconiosis; increased markings noted in all of the lung fields
(same)	DX 44	Scott, BCR, B	Negative for pneumoconiosis
(same)	DX 44	Wheeler, BCR, B	Negative for pneumoconiosis; few tiny scars
2/8/2000	DX 64	Shusterman	No pneumonia; no acute cardiopulmonary disease process
2/9/2000	DX 64	Kowalski	Lungs essentially clear
2/10/2000	DX 64	Schroeder	Lungs clear
2/11/2000	DX 64	Schroeder	Lungs clear
2/13/2000	DX 64	Mewborne	No focal airspace disease or interstitial edema
4/13/2000	DX 77	McMurray	Lungs are clear, no acute disease

Discussion

Of the twenty-three chest x-rays in presented in the record, almost all of the films (twenty-two) did not generate a dispute among the reviewing physicians. As a result, the chest x-rays of July 10, 1973, April 30, 1979, May 23, 1979, June 3, 1979, June 4, 1979, June 12, 1979, August 18,

1979, August 21, 1979, May 26, 1981, August 23, 1996, May 6, 1997, September 28, 1997, September 4, 1998, February 11, 1999, September 27, 1999, and December 8, 1999, February 8, 2000, February 9, 2000, February 10, 2000, February 11, 2000, February 13, 2000, and April 13, 2000 did not produce evidence of pneumoconiosis.

In fact, the medical experts disagreed only on the one remaining film of May 27, 1999. Dr. Paranthaman and Dr. Gaziano found sufficient evidence of pneumoconiosis in the film. However, their opinions are outweighed by the consensus of the two better qualified physicians, Dr. Scott and Dr. Wheeler, who are qualified as both B readers and board certified radiologists, that the film of May 27, 1999 is negative. Accordingly, since all of the radiographic evidence is negative, Mrs. Owens is unable to establish the presence of black lung disease in her husband's lungs through chest x-rays.

Although Mrs. Owens is unable to prove the presence of black lung disease in her husband's lungs by chest x-rays, she still may prevail on this element of entitlement if either biopsy or autopsy evidence supports a finding of pneumoconiosis.

Biopsy/Autopsy

(Note: the following summary, and other remaining portions of this decision, contain detailed information obtained from the autopsy of Mr. Owens, submitted to support his claim and Mrs. Owens' survivor claim. While respecting the dignity and privacy of the deceased, some discussion of the detailed observations is necessary because I find the medical information relevant on determining whether Mr. Owens had pneumoconiosis.)

Prior to reviewing the diverse reports on the examination of Mr. Owens' lung tissue, a review of the regulatory provisions on the requisite standard for diagnosing pneumoconiosis based on a biopsy or autopsy helps to understand the significance of some of the reports. The regulations define "clinical" pneumoconiosis as a condition characterized by permanent deposition of substantial amounts of particulate matter, caused by coal dust exposure, in the lungs **and** "the fibrotic reaction of the lung tissue to that deposition," 20 C.F.R. § 718.201 (a) (1) (emphasis added). Such reaction may be characterized as coal workers' pneumoconiosis, anthracosilicosis, anthracosis, anthrosilicosis, massive pulmonary fibrosis, silicosis, or silictuberculosis," 20 C.F.R. § 718.201 (a) (1). As a result, an autopsy or biopsy finding of anthracotic pigmentation, standing alone, is not sufficient to establish the presence of pneumoconiosis, 20 C.F.R. § 718.202 (a) (2).

Dr. Larry W. Joyce
(DX 58 and DX 68)

On September 19, 2000, Dr. Larry W. Joyce**,** board certified in anatomic and clinical pathology,[13] conducted an autopsy examination of Mr. Owens' chest area. Although he was not aware of Mr. Owens' medical background, Dr. Joyce was informed that Mr. Owens had been a coal miner for 25 to 30 years and smoked 3 to 4 cigarettes a day for 40 to 50 years.

During gross examination of the lungs, Dr Joyce observed that the mediastinal lymph nodes were "tan to mildly blackish." He bisected the largest node and found "some interspersed blackish foci measuring up to 1 cm in greatest dimension." Subcarinal lymph nodes were "brownish-black" and the peribronchial lymph nodes were "blackish." Turning to the lung surfaces, Dr. Joyce commented that the upper lobes were "mildly to moderately streaked with black pigment;" whereas, the right middle lobe and the lower lobes were "mildly streaked with black pigment." Additionally, Dr. Joyce commented "there are a few scattered blackish foci measuring up to 5 mm in greatest dimension scattered throughout the lower lobes and the right middle lobe and a moderate number of these foci are seen throughout the upper lobes." At the conclusion of his gross examination, Dr. Joyce's provisional diagnosis was coal workers' pneumoconiosis.

As part of the autopsy, Dr. Joyce obtained tissue samples from the mediastinal, subcarnial, peribronchial lymph nodes for microscopic examination. He also prepared pathology slides of lung tissue from the right upper, right middle, right lower, left upper, and left lower lobes.

Under the microscopic, Dr. Joyce saw in the lymph nodes "finely granular to slightly coarse, black pigment deposits. . . and one small silicotic nodule." His observations of the lung lobe tissue samples produced the following comments:

> Moderate to marked degree of pulmonary emphysema. . .[S]cattered foci of loose fibroconnective tissue which shows some intermixed macrophages showing rather heavy, finely granular to slightly coarse black pigment deposition within the cytoplasm. Surrounding these foci are emphysematous changes and the foci are consistent with a mild degree of coal workers' pneumoconiosis (coal macules).

In light of his microscopic findings, Dr. Joyce reached a final diagnosis of moderate to marked pulmonary emphysema, coal workers' pneumoconiosis "mild, coal (dust) macules," acute bronchopneumonia, and marked black pigment deposits in various lymph nodes.

Dr. Echols A. Hansbarger, Jr
(DX 62, DX 69, and DX 70)

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[13]I take judicial notice of Dr. Joyce's board certification and have attached the certification documentation. Since I was unable to advise the parties at a hearing of my intention to take judicial notice of the qualifications of physicians in this case, any party who objects to such judicial notice must enter a written objection within 10 days of the date of this decision, requesting my reconsideration of Dr. Joyce's medical qualifications.

On December 26, 2000, Dr. Echols A. Hansbarger, Jr., board certified in anatomic and clinical pathology, examined both the autopsy report and the 20 autopsy slides of which 17 are lung slides.

Upon microscopic examination, Dr. Hansbarger found moderate emphysematous changes with enlarged airspaces, thickened bronchi and moderate thickening of the bronchial walls. Extensive congestion was noted. A focal area of acute bronchopneumonia was noted. Scattered throughout the lung parenchyma were focal depositions of anthracotic pigment without reactive fibrosis. Dr. Hansbarger specifically did not see coal maculae. Pigment deposition was also noted on the bronchial lymph nodes.
The finding of anthracotic pigmentation of the lung and bronchial lymph nodes was not sufficient to warrant the diagnosis of coal workers' pneumoconiosis or any other occupational pneumoconiosis of the lung. Dr. Hansbarger diagnosed acute bronchopneumonia, moderate centrilobular emphysema, and severe lung congestion.

Dr. Joseph F. Tomashefski, Jr.
(DX 63 and DX 70)

On January 16, 2001, Dr. Tomashefski, board certified in anatomic and clinical pathology, reviewed the death certificate, autopsy protocol and personally examined the microscopic slides. His observations revealed diffuse airspace enlargement with centriacinar accentuation, and "apparently detached" septa. There was minimal black pigment in perivascular tissue and alveolar walls, but no coal macules were identified. Reactive peribronchial lymph nodes contained sparse, small, hyaline nodules with black pigment and birefringent crystals.

Since he did not observe coal macules in his examination, Dr. Tomashefski opined Mr. Owens did not have pneumoconiosis. The "small silicotic nodules in [Mr. Owens'] peribronchial lymph nodes are evidence of silica exposure." However, because Dr. Tomashefski found "no silicotic nodules in Mr. Owens' lung tissue," the physician concluded Mr. Owens did not have pulmonary silicosis. Dr. Tomashefski diagnosed severe obstructive lung disease, mixed panacinar and centriacinar emphysema, chronic bronchitis, and patchy bronchopneumonia.

Discussion

Upon initial consideration, the preponderance of the expert pathology opinion, in the form of Dr. Hansbarger's and Dr. Tomashefski's opinions, doesn't support a finding of pneumoconiosis. However, with further reflection and concentration on three aspects of the pathology reports, and the associated areas of the lungs, I ultimately conclude that the preponderance of the more probative pathology evidence does indeed support a finding of coal workers' pneumoconiosis.

The first area of attention is Dr. Joyce's gross examination of the lungs. Upon gross examination, Dr. Joyce first reported streaks of pigmentation, which as noted above do not support a finding of pneumoconiosis. However, he also dissected the largest mediastinal lymph node and

found a 1 cm blackish foci. Dr. Joyce next reported a moderate number of blackish foci up to 5 mm in diameter scattered throughout the upper lobes of the lungs with a lesser distribution in the right middle and lower lobes. Based on the presence of blackish foci, or masses, both in the mediastinal lymph node and a substantial portion of the lungs lobes, Dr. Joyce apparently found sufficient lung tissue reaction to provisionally diagnose coal workers' pneumoconiosis.

Notably, while the other two pathologists, Dr. Hansbarger and Dr. Tomashefski, reviewed Dr. Joyce's autopsy report, neither doctor addressed his observation of black foci upon gross examination. Perhaps, Dr. Hansbarger and Dr. Tomashefski believed their microscopic findings impeached Dr. Joyce's gross exam observations. Yet, neither pathologist made such a statement and their inability to find coal macules in the lung tissues samples does not necessarily negate Dr. Joyce's report of black foci scattered throughout the lungs. Had Dr. Joyce reached a definitive diagnosis of pneumoconiosis at the conclusion of the gross examination, then his opinion would have outweighed the assessments of Dr. Hansbarger and Dr. Tomashefski. However, Dr. Joyce also clearly made his pneumoconiosis diagnosis at the conclusion the gross examination provisional, reflecting the need to microscopically confirm this diagnosis, which leads to the second area of focus.

When the three pathologists examined the same pathology slides, Dr. Joyce saw the requisite coal macules to support a final diagnosis of pneumoconiosis; Dr. Hansbarger and Dr. Tomashefski did not. Since all three doctors were well qualified pathologists, I have little means to differentiate the stark contrast of their observations. I simply find that the reports by Dr. Hansbarger and Dr. Tomashefski that no coal macules are present in the lung tissue outweigh the sole contrary finding by Dr. Joyce. Accordingly, I conclude that the lung tissue samples on the microscopic slides did not reveal the necessary lung tissue reaction to the apparent black pigmentation for a diagnosis of pneumoconiosis. Further, since Dr. Joyce did not render a final diagnosis of pneumoconiosis until after his microscopic exam and his microscopic observation conflicts with my finding that coal macules are not present in the lung tissue samples, his conclusion that Mr. Owens had pneumoconiosis loses probative value.

In other words, Dr. Joyce's provisional diagnosis of pneumoconiosis based on his gross examination comes apart because he apparently needed, and found, microscopic confirmation that the black foci were coal macules. However, Dr. Hansbarger and Dr. Tomashefski did not see coal macules during the microscopic exam of samples from the lung areas containing the foci. As a result, Dr. Joyce's gross observation of blackish foci, and corresponding provisional black lung diagnosis, lacks microscopic confirmation and becomes, absence any further explanation by Dr. Joyce, an ambivalent finding that does not support a conclusion that Mr. Owens had pneumoconiosis.

While the first two areas of concentration do not support a finding of black lung disease, a third focus on the findings relating to the lungs' lymph nodes leads to a different conclusion. Dr. Joyce reported a microscopic finding that one of the lungs' lymph nodes contained a small silicotic nodule. In this case, while Dr. Hansbarger did provide any specific finding one way or the other, Dr. Tomashefski concurred with Dr. Joyce's observation by noting small silicotic nodules in the

peribronchial lymph nodes demonstrating exposure to silica. Consequently, based on the observations of Dr. Joyce and Dr. Tomashefski, I find that the lymph nodes associated with Mr. Owens' lungs contained evidence of the tissue reaction to silica exposure.

After commenting on the presence of silica nodules, Dr. Tomashefski proceeded to explain that since silica nodules were not also present in the lung tissue itself, a diagnosis of silicosis was not appropriate. Yet, Dr. Tomashefski's apparent requirement for finding nodules also in the lung tissue is contrary to legal precedent. In *Daugherty v. Dean Jones Coal Co.*, 895 F.2d 130 (4th Cir. 1989), the court rejected a pathologist's position that a biopsy of a lung lymph node showing anthracosis was insufficient to diagnose pneumoconiosis. The court observed that in *Bueno v. Director, O.W.C.P.*, 7 BLR 1-337, 1-340 (1984), the Benefits Review Board ("BRB") had determined a finding of anthracosis of the hilar lymph nodes may be sufficient, by itself, to support a finding of pneumoconiosis. *Daugherty*, 895 F.2d at 132. Once the requirement that silica nodules also be present in the lung tissue for a silicosis diagnosis is removed by the BRB and a court of appeals, Dr. Joyce' observation and Dr. Tomashefski's observation and statement may be read to support a conclusion that the lymphatic silica nodules in Mr. Owens' chest represent silicosis. Since the regulatory definition of pneumoconiosis, 20 C.F.R. § 718.201 (a) (1) specifically includes silicosis, I find the biopsy of his lungs' lymph nodes establishes that Mr. Owens had pneumoconiosis.

Compton Analysis

Although I have ultimately found that the biopsy evidence of the lymph nodes is sufficient to support a finding of pneumoconiosis, the court in *Compton* holds that I must further consider the autopsy and biopsy evidence in conjunction with both the radiographic evidence and the other medical opinions in the record to determine whether Mr. Owens has pneumoconiosis.

Turning to the chest x-rays, I have concluded the radiographic evidence is negative for the presence of pneumoconiosis. However, considering the small amount of silica nodules and the absence of widespread distribution, the failure of the x-rays to identify the silicosis is understandable. As a result, I do not believe the negative radiographic evidence negates the biopsy finding of pneumoconiosis.

Finally, I must review the extensive medical opinion in the record. While the post-mortem medical opinions are better documented than the earlier assessments, I will now summarize all the medical opinion which may have some relevance in the later portions of my analysis.

Medical Opinions

Dr. T.L. Wright
(DX 46-16 and DX 46-18)

On February 19, 1979, Dr. T.L. Wright examined Mr. Owens for pneumoconiosis on behalf of DOL. At that time, Mr. Owens reportedly smoked ½ pack of cigarettes per day for 20 years. The physical examination was unremarkable. The lungs were clear to auscultation and percussion. Mr. Owens was able to elevate his $_{P}O_{2}$ with exercise indicating normal ventilatory reserve. The chest x-ray was interpreted as normal. Based on Mr. Owens' physical examination and test results, Dr. Wright diagnosed hypertensive cardiovascular disease. There was no evidence of occupational pulmonary disease. Mr. Owens was, from a pulmonary standpoint, capable of continuing at his present occupation.

Dr. Michael J. Winsor
(DX 46-16)

In May and June of 1979, Dr. Winsor hospitalized Mr. Owens a couple of times for treatment of an acute illness caused by a fever with an unknown etiology. During his treatment, Mr. Owens informed Dr. Winsor that he had been diagnosed with black lung disease but continued to work in the coal mines. During physical examinations, Mr. Owens' chest was clear and a lung scans showed only a few perfusion defects compatible with COPD (chronic obstructive pulmonary disease). Later in the summer, around August 18, Mr. Owens returned to the hospital for treatment of acute bronchitis and pneumonia.

Dr. Dale E. Solomon
(DX 46-17and DX 46-19)

Between March and June 1980, Mr. Owens was re-hospitalized for acute right upper lobe pneumonia. After treatment, Dr Solomon conducted a series of pulmonary and respiratory tests. Based on the results of these studies, including a treadmill exercise test, Dr. Solomon noted Mr. Owens had low exercise tolerance. Dr. Solomon believed Mr. Owens struggled with two disease processes. First, Mr. Owens has a severe chronic obstructive disease that was a major disabling factor. Second, he also had a neuromuscular disorder of an unknown cause. According to Dr. Solomon, the combination of these two health problems totally disabled Mr. Owens.

Dr. James Tcheng and Dr. Richard Stack
(DX 44)

For ten days in July 1996, Mr. Owens was treated by Dr. Tcheng and evaluated by Dr. Stack for unstable angina. His prior medical history included a heart artery bypass graft in 1984 with three subsequent myocardial infarctions, with the latest occurring the month before his admission. Upon examination, the doctors heard wheezing breath sounds. Eventually, an angioplasty was performed which relieved some of the angina. However, Mr. Owens suffered pulmonary hemorrhage secondary to pneumonia. In addition to unstable angina, the physicians also diagnosed severe COPD, coronary artery disease, and hypertension.

Dr. G.S. Kanwal
(DX 47-8, DX 47-9, and DX 47-10)

On May 6, 1997, Dr. G.S. Kanwal examined Mr. Owens for pneumoconiosis. Mr. Owens reported coal mine experience for 28 years, mostly all of it inside the mines as a motorman, loading coal, and a past medical history of cardiac bypass surgery. He gave his smoking history as three cigarettes per day for ten years. He complained about wheezing all the time, shortness of breath at rest, occasional cough and frequent paroxysmal nocturnal dyspnea. He was also markedly limited and had difficulty walking. The chest x-ray was negative and the blood gas study showed mild hypoxemia. Due to his heart problems, Mr. Owens could not take the pulmonary function test. Dr. Kanwal diagnosed Mr. Owens as totally and permanently disabled due to cardiac condition and asthma. However, he found no evidence of pneumoconiosis.

Dr. S.K. Paranthaman
(DX 7, DX 10, and DX 11)

On May 27, 1999, Dr. S.K. Paranthaman evaluated Mr. Owens for pneumoconiosis. At this time, Mr. Owens gave his past employment history of approximately 30 years as a coal miner, all underground, with his last coal mine employment as a foreman. He quit work in 1984 due to health problems.

At the time of the evaluation, Mr. Owens reportedly smoked a third of a pack of unfiltered cigarettes per day for 25 years. He complained of a mild productive cough for 20 years, mostly in the morning hours and experienced wheezing for the past 8 years. He had hemoptysis about 6 months before the examination and was hospitalized for 2-3 days. Mr. Owens also suffered with bilateral anterior chest pain for 13 years that was relieved by medication.

Physical examination revealed that Mr. Owens was dyspneic at rest, while mild cyanosis was noted when he breathed room air. The chest showed an increase in AP diameter and breath sounds were decreased bilaterally. Inspiratory and expiratory wheezing was noted bilaterally. Heart sounds were masked by respiratory wheezing.

Mr. Owens' chest x-ray indicated the presence of pneumoconiosis. The pulmonary function tests revealed a severe airway obstruction[14] and the blood gas study indicated CO_2 retention and hypoxemia of moderate degree. Mr. Owens did not wish to take the exercise test due to heart and lung problems.

Based his examination, Dr. Paranthaman diagnosed simple coal workers' pneumoconiosis related to coal dust exposure for 30 years in the underground mines, if documented. He also found pulmonary emphysema and chronic bronchitis due to the combined effect of cigarette smoking for 25 years and coal dust exposure for 30 years, plus arteriosclerotic heart disease unrelated to coal dust exposure. In addition, on the basis of the pulmonary and blood gas tests, Dr. Paranthaman concluded Mr. Owens was totally disabled due his respiratory problems and thus unable to return to his former coal mine employment as a foreman.

[14]Dr. Ranavaya opined that the pulmonary function tests were invalid due to less than optimal effort.

Dr. Gregory J. Fino
(DX 37, DX 50, EX 3 and EX 4)

On December 8, 1999, Dr. Gregory J. Fino, board certified in internal medicine and pulmonary disease, examined Mr. Owens and summarized his findings in an initial report dated January 7, 2000. At the time of the evaluation, Mr. Owens was 70 years old and smoked ½ pack of cigarettes per day for 49 years, from 1949 until 5 months prior to the examination. His coal mine employment covered 30 years. Mr. Owens' breathing problems, characterized by shortness of breath, had been present for the last 25 years and were getting worse.

During the examination, Mr. Owens appeared chronically ill and in no acute distress. He was using supplemental oxygen and had a marked decrease in the breath sounds with a prolongation of the expiratory phase. Dr. Fino observed that Mr. Owens became dyspneic when walking at his own pace on level ground or ascending one flight of steps. Mr. Owens complained of chest pain, admitted to daily cough and mucous production, and wheezed.

The chest x-rays were classified as 0/0. Although increased markings appeared in all of the lung fields, there were no pleural and no parenchymal abnormalities consistent with an occupationally acquired pneumoconiosis. Pulmonary function studies were declined by the patient based on a letter from his physician. Oxygen saturation and carboxyhemoglobin level were normal. The arterial blood gas study showed significant hypoxia and hypercarbia.

In addition to the physical exam, Dr. Fino conducted a review of the medical record associated with Mr. Owens' previous applications for benefits and concluded Mr. Owens did not suffer from an occupationally acquired pulmonary condition as a result of coal mine dust exposure. The majority of chest x-ray readings were negative for pneumoconiosis. The acceptable pulmonary function tests were normal with no obstruction, restriction, or ventilatory impairment.[15] While Mr. Owens had a rapid change in his chest film over the last couple of years, such a change was not consistent with pneumoconiosis.

From a respiratory standpoint, his pulmonary system was abnormal such that Mr. Owens could not perform his last coal mining job due to significant hypoxia and hypercarbia that developed after 1997. His change was not related to coal dust exposure because, as Dr. Fino explains it, pneumoconiosis is not a latent condition. Citing medical studies, Dr. Fino observed that if a miner, such as Mr. Owens, left the mines with a negative x-ray film and there was no further dust exposure, the chance of developing coal workers' pneumoconiosis was zero.[16]

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[15]Due to the patient's poor effort, Dr. Fino found the pulmonary function tests performed by Dr. Kanwal on May 6, 1997 and Dr. Paranthaman on May 27, 1999 invalid because the spirometric and MVV values underestimated Mr. Owens' true lung function.

[16]Although Dr. Fino may be correct in terms of "clinical" pneumoconiosis, the regulations, in defining both clinical and legal pneumoconiosis, 20 C.F.R. § 718.201(c), clearly states that "pneumoconiosis' is recognized as a latent and progressive disease which may first become detectable only after the cessation of coal mine dust

(continued...)

In summary, Dr. Fino reasoned there was insufficient objective medical evidence to justify a diagnosis of simple coal workers' pneumoconiosis. Mr. Owens did not suffer from an occupationally acquired pulmonary condition. Although there was a disabling respiratory impairment present, that ailment was due to non-coal mine dust induced disease. From a respiratory standpoint, Mr. Owens was totally disabled from returning to his last mining job or a job requiring similar effort. Despite the material change in Mr. Owens' condition since 1997, this change would have occurred had he never stepped foot in the mines.

On April 4, 2000, Dr. Gregory J. Fino reviewed the medical record from Mr. Owens' hospitalization in July 1996, noting the discharge diagnoses included unstable angina, coronary artery disease, chronic obstructive pulmonary disease, hypertension, and hemoptysis secondary to possible pneumonia with pulmonary hemorrhage. Dr. Fino concluded this additional information did not alter his opinion on Mr. Owens' pulmonary condition and its cause.

On June 4, 2002, Dr. Fino further reviewed the hospital admissions that took place from January 1996 through September 2000 during Mr. Owens' last few years of life.[17] His last hospitalization occurred on September 12, 2000 for an emergency room treatment. Numerous physicians diagnosed chronic obstructive pulmonary disease. A summary of pulmonary function and arterial blood gas studies showed combinations of significant hypoxemia and hypercarbia. According to Dr. Fino, the development of Mr. Owens' lung disease occurred subsequent to the mid-1990s and its clinical course points to a non-coal mine dust related lung disease. A review of the newly submitted evidence did not alter Dr. Fino's prior conclusion that Mr. Owens' lung disease was related to diffuse pulmonary fibrosis, which is a disease of the general medical population. Furthermore, based on the objective medical evidence, and assuming Mr. Owens' death was respiratory, coal mine dust inhalation played no role in that death. Coal mine dust inhalation did not hasten his death; he would have died when he did even if he had never stepped foot in the mines.

Pit County Hospital, Greenville, South Carolina
(DX 64)

On February 8, 2000, Mr. Owens was admitted to the Pit County Hospital for respiratory distress. His reported medical history included COPD, congestive heart failure, coronary artery disease, black lung and asthma. Several doctors treated Mr. Owens for ten days. During this period,

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[16](...continued)
exposure.

[17]The patient had a series of hospital admissions which Dr. Fino reviewed in his supplemental report: 1/1/96 - 1/5/96; 4/22/96 - 4/26/96; 6/6/96 - 6/7/96; 6/25/96 - 7/2/96; 8/23/96 - 8/26/96; 11/21/96 - 12/4/96; 12/9/96 - 12/12/96; 1/9/97 - 1/14/97; 6/4/97 - 6/11/97; 6/14/97 - 6/19/97; 6/23/97 - 6/27/97; 9/22/97 - 9/24/97; 9/30/97 - 10/8/97; 11/4/97 - 11/11/97; 11/11/97 - 11/18/97; 1/12/98 - 1/16/98; 4/14/98 - 4/22/98; 6/14/98 - 6/18/98; 8/16/98 - 8/17/98; 9/4/98 - 9/8/94; 10/26/98 - 10/31/98; 12/11/98 - 12/15/98; 12/29/98 - 1/5/98; 1/15/99 - 1/29/99; 3/9/99 - 3/12/99; 7/30/99 - 8/4/99; 9/23/99 - 10/4/99; 10/22/99 - 10/30/99; 12/27/99 - 1/3/00; 1/21/00 - 1/28/00; 2/8/00 - 2/18/00; 4/7/00 - 4/14/00; 7/20/00 - 7/25/00; 8/18/00 - 8/22/00; and 8/28/00 - 9/1/00.

a CT chest scan showed patchy densities in both lungs and reticular nodules. The discharge diagnosis included COPD, CAD, aspiration pneumonia. His prognosis was guarded.

Holston Valley Medical Center
(DX 77)

On April 7, 2000, Dr. Sarrouji, Mr. Owens' treating physician,[18] referred Mr. Owens to the Holston Valley Medical Center for evaluation of a myocardial infarction. During the next week, Mr. Owens received treatment to relieve his angina and underwent a battery of the tests. Mr. Owens continued to complain about severe dyspnea upon exertion and a physical examination disclosed decreased breath sounds with distant wheezing and rhonchi. A chest x-ray was clear. The diagnosis included myocardial infarction, coronary artery disease, severe COPD, and history of tobacco abuse.

Dr. Bassam Sarrouji
(DX 58 and DX 68)

On September 29, 2000, Dr. Sarrouji signed Mr. Owens' death certificate listing the immediate cause of death as severe COPD. He also listed as an underlying cause CAD (coronary artery disease). Dr. Sarrouji did not indicate on the form whether he was aware of the September 19, 2000 autopsy results.

Dr. Larry W. Joyce
(DX 58 and DX 68)

In an October 6, 2000 letter, Dr. Joyce stated the autopsy examination of the lungs demonstrated a mild degree of coal workers' pneumoconiosis ("black lung"), moderate to severe emphysema, and some pneumonia in the right lung.

Dr. Echols A. Hansbarger, Jr
(DX 62, DX 69, and DX 70)

Based on his December 2000 examination of the pathology slides and review of the autopsy report, Dr. Hansbarger opined that Mr. Owens died on September 17, 2000 as a result of acute bronchopneumonia, chronic obstructive pulmonary disease and coronary artery disease, as listed on the death certificate. He found insufficient evidence for a diagnosis of pneumoconiosis. As a result, pneumoconiosis did not contribute to his death. Further, Mr. Owens had no respiratory impairment or pulmonary disability due to his coal mine employment.

Dr. Joseph F. Tomashefski, Jr.

[18] *See* admission notation, dated April 7, 2000 (DX 77)

(DX 63 and DX 70)

In addition to his review of the autopsy slides, Dr. Joseph F. Tomashefski, Jr. in January 2001 also reviewed Mr. Owens' death certificate, autopsy report, autopsy slides and partial medical history, including chest x-rays, pulmonary function tests, arterial blood gas studies and physical examinations. After observing Mr. Owens' history of approximately 30 years of coal mine employment and 50 years of cigarette smoking, and reviewing his medical history, Dr. Tomashefski found Mr. Owens' health condition significant for severe coronary artery disease (including angioplasty, stint replacement, and coronary by-pass graphs), and episodes of congestive heart failure. Mr. Owens had also experienced three previous myocardial infarcts, and had a history of hypertension. In 1996, a coronary angiogram demonstrated 95% stenosis of the right coronary artery and left circumflex artery, and proximal occlusion of the left anterior descending artery. In 1996, he underwent a coronary angioplasty with stenting of the left circumflex and right coronary arteries. According to the medical report of May 27, 1999, Mr. Owens was taking nitroglycerin for chest pain and he complained of two pillow orthopnea, ankle swelling and nocturnal dyspnea.

From a respiratory standpoint, Dr. Tomashefski reported that Mr. Owens complained of wheezing, sputum production, cough and dyspnea. His medications included prednisone, bronchodilators and inhaled steroids for a clinical diagnosis of chronic obstructive pulmonary disease. On physical examination in May 1999, Mr. Owens was noted to be cyanotic, with digital clubbing and an increased AP chest diameter. His chest was hyperresonant to percussion, and he had bilaterally decreased breath sounds, and inspiratory and expiratory wheezing. He had been previously hospitalized for an acute exacerbation of COPD and ill-defined febrile illnesses. Chest x-ray interpretations indicated the presence of calcified granulomas, and hyperinflation consistent with emphysema. The majority of reports were also negative for pneumoconiosis. The results of Mr. Owens' serial pulmonary function tests were compatible with obstructive lung disease with little reversibility after bronchodilator administration. Mr. Owens' most recent arterial blood gas study indicated severe hypoxemia ($_pO_2$ 50 mm Hg) and hypercarbia ($_pCO_2$ 64 mm Hg). Further, Dr. Tomashefski explained that neither coal mine employment nor coal dust exposure was a cause of mixed panacinar/centriacinar emphysema, chronic bronchitis which persists after coal dust exposure ceases, small airways disease, aspiration pneumonia or coronary artery disease. Whereas, cigarette smoking is an important cause of emphysema, chronic bronchitis and small airways disease, and it is also an important risk factor for coronary disease. Thus, Dr. Tomashefski concluded that Mr. Owens' death and respiratory symptoms were unrelated to his work as a coal miner. In the absence of medical records contemporaneous with Mr. Owens' death, Dr. Tomashefski was unable to determine the cause of death. However, at the time of his death, Mr. Owens had moderately severe obstructive lung disease characterized by mixed panacinar and centriacinar emphysema, chronic bronchitis, and small airways disease.

Dr. Peter G. Tuteur
(DX 62, DX 69, EX 1, and EX 2)

On June 9, 2000, three months before Mr. Owens passed away, Dr. Peter G. Tuteur, board certified in internal medicine and pulmonary disease, conducted an independent medical review of

Mr. Owens' records from February 1979 through December1999, including a vast array of chest x-ray interpretations, pulmonary and respiratory tests and examination and hospitalization records. For 30 years, Mr. Owens worked as an underground coal miner running a motor, loading coal, and eventually serving as a foreman before he retired in 1984 because of health reasons. In addition, he smoked cigarettes for nearly 50 years. Quantification of this activity ranged between 1/3 and one package a day, putting Mr. Owens at risk for the development of tobacco smoke associated heath problems including chronic obstructive pulmonary disease (chronic bronchitis/emphysema), arteriosclerotic heart disease, and/or lung cancer.

Dr. Tuteur observed Mr. Owens' cardiovascular history included advanced coronary artery disease, impacting on left ventricular function treated both with aggressive medical management and surgical treatment including CABG (coronary artery bypass graft), angioplasty, and stint placement. Mr. Owens' condition was further complicated by a personal and family history of hypertension.

Mr. Owens' pulmonary history began with "asthma as a child." Details concerning this notation were not available; however, Dr. Tuteur noted that childhood pulmonary disease such as asthma substantially increases the risk for airways obstruction as an adult. During his work years, Mr. Owens experienced increasingly severe breathlessness and eventually was treated with a regimen typically used for airways obstructions. His respiratory condition was complicated by episodes of sinusitis (best documented in May, 1979 with CT scan and response to therapy) and recurrent pneumonias sometimes requiring hospitalization on five occasions between 1994 and 1999. Wheezing was frequent as was recurrent chest pain interpreted as angina.

Physical examinations of the chest waxed and waned between normal and signs of airways obstruction in the form of decreased intensity of breath sounds, prolongation of expiration, and intermittent wheezing. Pulmonary function studies were conducted over time. Unfortunately, these studies were not always valid as an assessment of maximum function. The initial studies of 1973 were within normal limits. Arterial blood gas analysis, both at rest and during exercise, were normal on two occasions in 1979. Thereafter, numerical data worsened demonstrating a progressive obstructive defect not associated with a restrictive component and the eventual development of hypoventilation (CO_2 retention) and resultant hypoxemia. Chest radiographs almost always were interpreted as free of changes compatible with coal workers' pneumoconiosis. Intermittently, infiltrative or nodular processes were identified only to change, sometimes resolve, over time.

Based on the record review, Dr. Tuteur opined Mr. Owens' pulmonary history, with risk factors of childhood illness and cigarette smoking and a clinical course (more rapidly progressive distant rather than proximal to his experience in the coal mines) associated with an obstructive but not a restrictive abnormality and characterized by recurrent pulmonary infections and/or hemorrhages, was "quintessentially typical" of that of cigarette smoke-induced chronic obstructive pulmonary disease. It in no way resembled the clinical picture expected in a person with coal workers' pneumoconiosis.

Mr. Owens was totally disabled due to: a) cardiopulmonary disease in the form of advanced coronary artery disease incompletely responsive to both surgical and medical management; and, b)

advanced cigarette smoke-induced chronic obstructive pulmonary disease manifested both by emphysema and chronic bronchitis. These conditions were in no way related to, aggravated by, or caused by the inhalation of coal mine dust or the development of coal workers' pneumoconiosis.

Unfortunately, Mr. Owens' breathlessness and exercise intolerance worsened over time. Although a quintessential feature of clinically significant simple coal workers' pneumoconiosis, such a problem was also consistent with virtually any primary pulmonary and/or cardiac disorder. In this case, Mr. Owens had coronary artery disease resulting in angina pectoris, congestive heart failure, exercise intolerance, all despite aggressive medical and surgical management. In addition, he clearly had obstructive lung disease not only manifested by breathlessness, but also by cough, expectoration, and wheezing. Yet, cough, expectoration, wheezing and chest pain are not regular features of coal workers' pneumoconiosis.

Moreover, Dr. Tuteur observed that physical examination of Mr. Owens' chest varied over time. On occasions it was reported as well within normal limits. At other times, signs of airways obstruction of varying degree were demonstrated in the form of decreased intensity of breath sounds, prolongation of expiration as well as wheezing. These findings were typical of airways obstruction such as occurs in cigarette smoke-induced chronic obstructive pulmonary disease. In contrast, when coal workers' pneumoconiosis is sufficiently advanced to produce abnormalities on physical examination, one expects to find decreased lung expansion and/or the presence of persistent late inspiratory crackling sounds.

In the numerical values associated with pulmonary function testing from 1993 to present, Dr. Tuteur observed a progressive obstructive ventilatory defect (assuming the numerical data are valid) from a point in 1973, when the values were well within normal limits, to the severe abnormality on the most contemporaneously relevant studies. This obstructive defect was not associated with a restrictive component. Initially and through 1979, arterial blood gas analysis was well within normal limits both at rest and during exercise. Subsequently, gas exchange became impaired and alveolar hypoventilation (CO_2 retention) developed. These findings were typical of the progression of cigarette smoke-induced chronic obstructive pulmonary disease. He further noted that the most rapid progression developed long after the cessation of coal mine dust exposure. In contrast, when coal workers' pneumoconiosis is sufficiently advanced to produce impairment of pulmonary function, one expects not an obstructive ventilatory defect (though it may occur under rare circumstance) but one does expect a restrictive ventilatory defect manifested by a reduced total lung capacity and/or impairment of gas exchange, first occurring during exercise, then at rest. Here, there was no restrictive component; gas exchange was relatively well preserved as alveolar hypoventilation developed and was responsible for the hypoxemia. Again, these changes were typical of advancing obstructive lung disease due to the chronic inhalation of tobacco smoke.

In addition, the overwhelming consensus of the chest x-rays indicated the absence of changes compatible with coal workers' pneumoconiosis, and the intermittent presence of changes which tended to resolve reflecting recurrent acute infiltrative processes clinically associated with exacerbation of COPD.

Dr. Tuteur concluded Mr. Owens did not have clinically significant, physiologically significant, or radiographically significant coal workers' pneumoconiosis or any other coal mine dust-induced disease process. However, Mr. Owens clearly was disabled to such an extent that he was unable to fulfill the requirements of a coal miner or work requiring similar effort. Dr. Tuteur stated that even if radiographic evidence of coal workers' pneumoconiosis existed, the disease would be of insufficient severity and profusion adversely affect Mr. Owens' pulmonary capacity.

On May 16, 2002, Dr. Tuteur accomplished a supplemental medical review of Mr. Owens' record including new medically relevant data. Again, the physician noted both 30 years of coal mine employment and 50 years of cigarette use. As a result, both coal dust and cigarette smoke represented pulmonary risks for Mr. Owens.

From a cardiac perspective, Mr. Owens had advanced coronary artery disease manifested by angina pectoris and acute myocardial infarctions and was treated with various forms of procedures, including angioplasty, stent placement, and coronary artery bypass grafts. Physiologically, electrocardiograms were abnormal because of right bundle branch block and other dysrhythmias and clinical and echocardiographic evidence of left ventricular dysfunction resulting in episodes of congestive heart failure.

In terms of pulmonary condition, Dr. Tuteur observed Mr. Owens experienced progressive breathlessness to a severe state eventually requiring maximal medical management in the form of inhaler, supplemental oxygen therapy, and systemic corticosteroids. Complicating the lower respiratory problems were recurrent episodes of sinusitis and recurrent documented pneumonias. In addition, he very frequently was admitted to the hospital for evaluation and management of acute exacerbations of chronic obstructive pulmonary disease. During the last 5 years of life, Mr. Owens was admitted to the hospital for such conditions for more than 35 times. On September 17, 2000, Mr. Owens died due to respiratory failure associated with severe COPD complicated by advanced coronary artery disease.

With respect to the etiology of his pulmonary problem, Mr. Owens' symptoms were characterized predominantly by severe breathlessness and exercise intolerance, associated with chronic daily productive cough and wheezing. The chest pain regularly experienced was almost certainly totally attributable to his coronary artery disease and resultant myocardial ischemia. Physical examination was marked with waxing and waning findings which, when abnormal, demonstrated airflow obstruction manifested by decreased intensity of breath sounds, prolongation of expiration, and intermittent adventitious sounds including wheezes. As indicated in the initial independent medical review, physiologic studies demonstrated progressive airflow obstruction from minimal to severe over the 2 ½ decades beginning in 1973 associated with normal total lung capacity measurements (i.e., no restriction) and progressive impairment of gas exchange as well as the interval development of at least intermittent CO_2 retention (alveolar hypoventilation). Risk factors for the development of such severe airflow obstruction, progressive even in the absence of exposure to coal mine dust but in the presence of continued cigarette smoking, include not only the cigarette smoking, but also the history of "childhood asthma." "Though coal mine dust exposure can produce airflow obstructions at times, such an occurrence, particularly the severity experienced by Mr. Owens, is quite

unusual in contrast to its occurrence in persons who chronically smoke cigarettes, but were never exposed to coal mine dust."

Additionally, Dr. Tuteur noted that even though Dr. Hansbarger saw evidence of pneumoconiosis, it was too minimal for a diagnosis of pneumoconiosis.[19] Even if the autopsy had established the presence of pneumoconiosis, it clearly was of an insufficient severity and profusion to produce clinical, physiological and radiographic evidence of the disease. In absence of such other manifestations, Dr. Tuteur concluded any pneumoconiosis that may have been present did not impact Mr. Owens' pulmonary physiology or contribute to his death.

Based on all available medical data, Dr. Tuteur opined Mr. Owens died with, and because of, severe and advanced pulmonary disease complicated by severe and advanced coronary artery disease, both resulting in lung and heart dysfunction, respectively. The etiology of the heart disease is coronary artery obstruction with risk factors including cigarette smoking and hyperlipidemia. The inhalation of coal mine dust played no role in the development of, the severity of, or the rate of progressive coronary artery disease in general, or in the coronary artery disease experienced by Mr. Owens. Nor did it hasten his death. Had Mr. Owens never worked in the coal mine industry, his clinical course, qualitatively, quantitatively, and chronologically, would have been no different.

Dr. John A. Michos
(DX 12 and DX 72)

On May 11, 2001, Dr. John A. Michos, board certified in internal and pulmonary medicine, conducted a medical review of Dr. Paranthaman's May 27, 1999 pulmonary examination of Mr. Owens, with the corresponding pulmonary function study, arterial blood gas tests, and x-ray reports. He also reviewed the death certificate, autopsy report, report from Dr. Peter Tuteur, report from Dr. Echols Hansbarger, review by Dr. Joseph Tomashefski, and medical reports from Pit County Medical Hospital. From the medical evidence provided, Dr. Michos concluded Mr. Owens did not have evidence of simple coal workers' pneumoconiosis, nor was his death hastened or caused by his prior coal mine employment.

Dr. Michos based his opinion upon autopsy reviews which either documented the absence of simple coal workers' pneumoconiosis or which described evidence for the presence of a mild simple coal workers' pneumoconiosis. Dr. Michos opined that this degree of simple coal workers' pneumoconiosis (if present) would not account for the severe degree of COPD described at autopsy.

[19]As noted in my review, Dr. Hansbarger reported the absence of both reactive tissue fibrosis and coal macules. Consequently, contrary to Dr. Tuteur's comment, Dr. Hansbarger did not find evidence of pneumoconiosis.

Instead, a significant, continuous history of tobacco abuse is well known to account for the type of disabling symptoms experienced by Mr. Owens. Mr. Owens' pulmonary condition in combination with his coronary artery disease and risks for aspiration, would probably account for his death and not his prior coal mine employment.

Discussion

Due to the dated nature of their examinations and conclusions, the opinions of Dr. Winsor, Dr. Wright, Dr. Solomon, Dr. Echeng, Dr. Stack, and Dr. Kanwal are not as well documented, or probative, as the more recent medical opinions of Dr. Paranthaman, Dr. Fino, Dr. Sarrouji, Dr. Joyce, Dr. Hansbarger, Dr. Tomashefski, Dr. Tuteur, and Dr. Michos. Of this later group of physicians, only Dr. Paranthaman and Dr. Joyce diagnosed pneumoconiosis.[20] Thus, the substantial majority of the medical opinion runs contrary to my finding of silicosis/pneumoconiosis based on the pathology findings of Dr. Joyce and Dr. Tomashefski. However, other than Dr. Tomashefski, none of the physicians who rendered an opinion that Mr. Owens did not have pneumoconiosis considered whether the presence of silica in Mr. Owens' pulmonary lymph nodes supported a diagnosis of pneumoconiosis. Instead, their negative assessments are due to the absence of radiographic evidence showing pneumoconiosis, focus on clinical pneumoconiosis, the lack of clinical and physiological evidence of pneumoconiosis and/or the negative pathology reports by Dr. Hansbarger and Dr. Tomashefski, which I have already determined do not legally outweigh the finding of lymphatic silicosis by Dr. Joyce and Dr. Tomashefski. Consequently, their opinions have little relevance on the issue of whether Mr. Owens has silicosis which leads to a diagnosis of pneumoconiosis. In the absence of any relevant contrary medical opinion on whether Mr. Owens had silicosis/pneumoconiosis, I conclude that a finding of silicosis/pneumoconiosis remains intact upon consideration of all the medical opinion in the record. Consequently, Mrs. Owens has established that the presence of pneumoconiosis in her husband's lungs.

Pneumoconiosis Arising Out of Coal Mine Employment

Having proven that her husband had pneumoconiosis, Mrs. Owens must next demonstrate that her husband's pneumoconiosis arose out of his coal mine employment. As indicated earlier, under the regulations, if a miner works ten or more years in one or more mines, a presumption exists that his or her pneumoconiosis arose out of coal mine employment. Since the record establishes that Mr. Owens mined coal for nearly thirty years, and in the absence of sufficient evidence to the contrary, notably any other silica exposure, I find his silicosis/pneumoconiosis arose out of his coal mine employment.

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[20]As previously discussed, due to absence of coal macules in the lung tissue slides, Dr. Joyce's diagnosis loses probative value. Based on a positive chest x-ray, Dr. Paranthaman rendered a diagnosis of clinical pneumoconiosis. However, his positive chest x-ray interpretation which is contrary to my finding that the radiographic evidence is negative for the presence of pneumoconiosis. Also , by linking Mr. Owens' emphysema to both cigarette smoke and coal dust, Dr. Paranthaman presented a diagnosis of legal pneumoconiosis. In this case, his opinion is more than offset by Dr. Michos' contrary opinion and Dr. Tuteur's in-depth explanation on how he determined coal dust was not a factor in Mr. Owens' obstructive defect.

Issue No. 2 - Death Due to Pneumoconiosis

With the first three elements of entitlement proven (eligible survivor, presence of pneumoconiosis, and pneumoconiosis arising out of coal mine employment), Mrs. Owens may receive survivor benefits if the preponderance of the evidence in the record establishes that her husband's death was due to pneumoconiosis. For a survivor claim filed on or after January 1, 1982, the Department of Labor regulations provide four means to establish that a coal miner's death was due to pneumoconiosis:[21]

1. The miner had complicated pneumoconiosis;[22]

2. Death was caused by pneumoconiosis;

3. Death was caused by complications of pneumoconiosis; or,

4. Pneumoconiosis was a substantially contributing cause or factor leading to the miner's death. Notably, pneumoconiosis is deemed to be a substantially contributing cause of a miner's death if it hastens the miner's death.[23]

However, a survivor may not receive benefits if the coal miner's death was caused by traumatic injury, or the principal cause of death was a medical condition not related to pneumoconiosis, unless evidence establishes that pneumoconiosis was a substantially contributing cause of death.

Complicated Pneumoconiosis

Under the regulations, 20 C.F.R. § 718.304, a massive lesion in the lungs that qualifies as complicated pneumoconiosis establishes an irrebuttable presumption that the coal miner's death was due to pneumoconiosis. Mr. Owen's radiographic and pathologic record does not establish the presence of such a large lesion or complicated pneumoconiosis. As a result, Mr. Owens did not have complicated pneumoconiosis and his surviving wife may not rely on this means of demonstrating that his death was due to pneumoconiosis.

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[21]20 C.F.R. §§ 718.205 (c)(1), (2), and (3), and 718.304.

[22]According to 20 C.F.R. § 718.304, if a miner had complicated pneumoconiosis, an irrebuttable presumption exists that his death was due to pneumoconiosis.

[23]20 C.F.R. §718.205 (c) (5). Previously, the U.S. Court of Appeals for the Fourth Circuit had adopted the U.S. Department of Labor's position that pneumoconiosis substantially contributes to death if it hastens death in any way. *Shuff v. Cedar Coal Co.*, 967 F.2d 977, 979 (4th Cir. 1992), *cert. denied,* 113 S.Ct. 969 (1993). *See also Lukosevicz v. Director, OWCP*, 888 F.2d 1001, 1006 (3d Cir. 1989) (any condition, such as pneumoconiosis, that hastens a coal miner's death is a "substantially contributing cause").

Death Caused By Pneumoconiosis

No physician in the record has indicated that pneumoconiosis caused Mr. Owens' death, even though the death certificate indicates he died due to respiratory failure. Notably, Mr. Owens' treating physician, Dr. Sarrouji, did not mention pneumoconiosis on the death certificate and the record contains no indication that Dr. Sarrouji considered Mr. Owens' severe COPD to be related, in part, to his exposure to coal dust. Additionally, since Dr. Joyce did not have access to Mr. Owens' medical records at the time of the autopsy, he declined to express an opinion on the cause of death. Finally, while Dr. Tuteur and Dr. Michos did not believe Mr. Owens had pneumoconiosis, they also opined that even if radiographic or pathology evidence of the disease were present, the minimal extent and profusion of the disease would have had no impact on the circumstances of his death. In light of the evidentiary insufficiency, I find Mr. Owens' death was <u>not</u> caused by pneumoconiosis.

Death Cause by Complications of Pneumoconiosis

In a similar manner, the record also precludes a finding the complications of pneumoconiosis caused Mr. Owens' demise. No physician linked pneumoconiosis, or its complications, to Mr. Owens' death. Even Dr. Joyce who found evidence of pneumoconiosis, characterized its severity as mild. Dr. Tuteur and Dr. Michos echoed that observation. Again, the later two physicians opined that the extent of pneumoconiosis, if present, was inadequate to have caused any effect on Mr. Owens' pulmonary condition.

Pneumoconiosis Was A Substantially Contributing Cause Of, Or Hastened, Death

A strong argument exists that when a miner has pneumoconiosis and experiences a respiratory death, black lung disease must have hastened the fatal event in some fashion. However, in Mr. Owens' case, no physician, including neither Dr. Sarrouji nor Dr. Joyce, has come forward and presented such a medical conclusion. Absent any medical opinion to support that supposition, I am unable to conclude that silicosis/pneumoconiosis hastened Mr. Owens' death.

Conclusion

Although Mrs. Rosalie D. Owens is an eligible survivor under the Act and her husband, Mr. Andrew C. Owens, had silicosis/pneumoconiosis due his coal mine employment, the record contains insufficient evidence to establish that his death was caused by pneumoconiosis, or as a consequence of coal workers' pneumoconiosis. Additionally, the medical record fails to prove that coal workers' pneumoconiosis substantially contributed to, or hastened his death. Accordingly, Mrs. Owens has failed to carry her burden of proof to establish that Mr. Owens' death was due to pneumoconiosis and her claim for survivor benefits under the Act must be denied.

Mr. Owens' Claim

Issue No. 3 - Material Change in Conditions

In 1997, Mr. Owens' second claim for benefits was denied due to his failure to prove either the presence of pneumoconiosis or a total respiratory impairment. Now, in his third, and duplicate, claim for benefits, Mr. Owens has, posthumously, demonstrated the presence of pneumoconiosis in his lungs. As a result, a material change in condition has occurred since the denial of his prior claim. Consequently, the entire record must be evaluated to determine whether Mr. Owens was entitled to disability compensation under the Act. *See* 20 C.F.R. § 725.309 (d).

Issue No. 4 - Black Lung Disability Entitlement

To receive compensation under the Act, a miner must prove four basic elements by a preponderance of the evidence. First, the miner must establish the presence of pneumoconiosis. Second, it must be determined whether the pneumoconiosis arose, at least in part, out of coal mine employment.[24] Third, the miner has to demonstrate he is totally disabled.[25] And, fourth, the miner must prove the total disability is due to pneumoconiosis.[26]

Presence of Coal Workers' Pneumoconiosis

The first two requisite elements have already been established. In my adjudication of Mrs. Owens' survivor claim, I have determined that Mr. Owens had silicosis/pneumoconiosis due to his coal mine employment.

Total Disability

The third necessary element for entitlement of benefits is total disability due to a respiratory impairment or pulmonary disease. If a coal miner suffers from complicated pneumoconiosis, there is an irrebuttable presumption of total disability. 20 C.F.R. §§ 718.204 (b) and 718.304. If that presumption does not apply, then according to the provisions of 20 C.F.R. § 718.204 (b) (2), in the absence of contrary evidence, total disability in a living miner's claim may be established by four methods: (i) pulmonary function tests; (ii) arterial blood-gas tests; (iii) a showing of cor pulmonale with right-sided, congestive heart failure; or (iv) a reasoned medical opinion demonstrating a coal miner, due to his pulmonary condition, is unable to return to his usual coal mine employment or engage in similar employment in the immediate area (20 C.F.R. § 718.204 (b) (1)).

While evaluating evidence regarding total disability, an administrative law judge must be cognizant of the fact that the total disability must be respiratory or pulmonary in nature. The U.S.

[24]20 C.F.R. § 718.203 (a).

[25]20 C.F.R. § 718.204 (b).

[26]20 C.F.R. § 718.204 (a).

Court of Appeal for the Third Circuit has held that, in order to establish total disability due to pneumoconiosis, a miner must first prove that he suffers from a respiratory impairment that is totally disabling, separate, and apart from other non-respiratory conditions.[27]

The record does not contain evidence of complicated pneumoconiosis or cor pulmonale with right-sided congestive heart failure.[28] Accordingly, Mr. Owens must demonstrate total respiratory or pulmonary disability through pulmonary function tests, arterial blood-gas tests, or medical opinion.

In that regard, the recent blood gas studies, in particular Dr. Paranthaman's May 27, 1999 test, showed Mr. Owens had a totally disabling respiratory impairment in the form of severe CO_2 retention and moderate hypoxemia.[29] Based on this test, Dr. Paranthaman, Dr. Fino, and Dr. Tuteur considered Mr. Owens totally disabled by his respiratory impairment. Additionally, Mr. Owens' multiple hospitalizations in the last year of his life document his severe respiratory distress. In light of the most recent medical data, I find Mr. Owens was totally disabled and unable to return to his coal mine employment due to a pulmonary impairment.

Total Disability Due to Pneumoconiosis

Because Mr. Owens has established three of the four requisite elements for entitlement to benefits, the award of black disability benefits rests on the determination of whether his respiratory disability is due to pneumoconiosis. Proof that a claimant has a totally disabling pulmonary disease does not, by itself, establish the impairment is due to pneumoconiosis. Absent regulatory presumptions in favor of the claimant,[30] the miner must under 20 C.F.R. § 718.204 (c) (1) demonstrate that pneumoconiosis was a substantially contributing cause of his total disability by showing the disease: 1) had a material, adverse effect on his respiratory or pulmonary condition; or, 2) materially worsened a totally disabling respiratory impairment caused by a disease for exposure unrelated to pneumoconiosis.

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[27]*See Beatty v. Danri Corp. & Triangle Enterprises and Dir., OWCP*, 49 F.3d 993 (3d Cir. 1995).

[28]While Mr. Owens had chronic coronary artery disease, none of the treating, examining or reviewing physicians indicated that cor pulmonale was involved.

[29]The resting arterial blood gas study (DX 11), which was verified as acceptable by Dr. Michos (DX 12) disclosed a $_pCO_2$ reading of 64, which establishes total respiratory impairment under the regulations. *See* Under 20 C.F.R. § 718.204 (b) (2) (ii).

[30]20 C.F.R. § 718.305 (if complicated pneumoconiosis is present, then there is an irrebuttable presumption the miner is totally disabled due to pneumoconiosis); 20 C.F.R. § 718.305 (for claims filed before January 1, 1982, if the miner has fifteen years or more coal mine employment, there is a rebuttable presumption that total disability is due to pneumoconiosis); and, 20 C.F.R. § 718.306 (a presumption when a survivor files a claim prior to June 30, 1982).

Since Mr. Owens did not have complicated pneumoconiosis and he filed his present claim after 1982, he would not be able to rely on any of the regulatory presumptions. Instead, Mr. Owens must use medical opinion in the record to establish that his total disability is due to pneumoconiosis. *See* 20 C.F.R. § 718.204 (c) (2). And, on the issue of total disability due to pneumoconiosis, the medical experts reached different conclusions. By diagnosing a total disability due to respiratory problem, evidenced by a severe obstruction, and opining coal dust exposure was a partial cause of Mr. Owens' pulmonary emphysema, Dr. Paranthaman essentially rendered an opinion that Mr. Owens was totally disabled due to pneumoconiosis. However, after an extensive medical record review, Dr. Tuteur reached a contrary opinion to the effect that even if Mr. Owens had pneumoconiosis, it was of minimal severity.

In light of this dispute, I must first assess the relative probative value of each medical opinion. The two factors I consider in evaluating relative probative weight are: a) documentation and b) reasoning.

As to the first factor, a physician's medical opinion is likely to be more comprehensive and probative if it is based on extensive objective medical documentation, such as chest x-rays, pulmonary function tests, arterial blood gas studies, and physical examinations. *Hoffman v. B & G Construction Co.*, 8 B.L.R. 1-65 (1985). In other words, a doctor who considers an array of medical documentation that is both long (involving comprehensive testing) and deep (includes both the most recent medical information and past medical tests) is in a better position to present a more probative assessment than the physician who bases a diagnosis on a test or two and one encounter.

The second factor of reasoning involves an evaluation of the connections a physician makes based on the documentation before him or her. A doctor's reasoning that is both supported by objective medical tests and consistent with all the documentation in the record, is entitled to greater probative weight . *Fields v. Island Creek Coal Co.*, 10 B.L.R. 1-19 (1987). Additionally, to be considered well reasoned, the physician's conclusion must be stated without equivocation or vagueness. *Justice v. Island Creek Coal Co.*, 11 B.L.R. 1-91 (1988).

With the two probative factors in mind, I find Dr. Paranthaman's medical opinion to have diminished relative probative weight principally due to a documentation problem. Since Dr. Paranthaman rendered his opinion in May 1999, he obviously did not consider the subsequent autopsy and pathology reports. Consequently, he rendered his medical opinion without considering the significant post-mortem evidence.

Dr. Paranthaman's shortfall in documentation is readily apparent, and adversely affects the probative value of his assessment, because the pathology evidence fails to support a connection between Mr. Owens' emphysema and his coal mine employment. After finding Mr. Owens totally disabled due to his respiratory problems, Dr. Paranthaman then simply stated that his emphysema was due to the "combined" effects of cigarette smoke and coal dust. However, although black streaks appeared in Mr. Owens' lungs, both Dr. Hansbarger and Dr. Tomashefski failed to find any lung tissue reaction to the pigmentation within the emphysematous tissue. Further, as Dr. Tuteur explained, the type of emphysema identified by the autopsy and pathology reports is not associated

with coal dust exposure. Thus, due to incomplete and out of date documentation, not only is Dr. Paranthaman's diagnosis of legal pneumoconiosis compromised, his conclusion that coal dust was a factor in Mr. Owens' total respiratory disability appears unsupportable. As a consequence, I consider Dr. Paranthaman's opinion, standing alone, insufficient to support a finding the Mr. Owens was totally disabled due to pneumoconiosis.

Further, in contrast, Dr. Tuteur presented a well documented and reasoned medical opinion, which is most consistent with all the medical evidence in the record including the preponderance of the pathology findings. Upon consideration of this extensive medical record, Dr. Tuteur explained that the results of the multiple examinations, chest x-rays, and pathology reports,[31] indicate the extent of any pneumoconiosis Mr. Owens may have had was so minimal as not to have affected his respiratory condition in any manner.

Conclusion

Mr. Owens had coal workers' pneumoconiosis and struggled in the last years of his life with a totally disabling pulmonary impairment. However, the better documented and more probative medical opinion does not support a finding that Mr. Owens' silicosis/pneumoconiosis contributed to his severe pulmonary condition. Accordingly, Mr. Owens' claim for black lung disability benefits under the Act must be denied.

ORDER

1. The claim of MRS. ROSALIE D. OWENS for survivor benefits under the Act is **DENIED**.

2. The claim of MR. ANDREW C. OWENS for disability benefits under the Act is **DENIED**.

SO ORDERED:

Richard T. Stansell-Gamm
RICHARD T. STANSELL-GAMM
Administrative Law Judge

Date Signed: December 9, 2002
Washington, DC

[31]I note even Dr. Joyce characterized the extent of pneumoconiosis that he observed as mild. Additionally, neither Dr. Joyce nor Dr Tomeshefski observed the spread of silicosis to the lungs' lobes.

NOTICE OF APPEAL RIGHTS: Pursuant to 20 C.F.R. § 725.481, any party dissatisfied with this Decision and Order may appeal it to the Benefits Review Board within 30 days from the date this decision is filed with the District Director, Office of Worker's Compensation Programs, by filing a notice of appeal with the Benefits Review Board, ATTN.: Clerk of the Board, Post Office Box 37601, Washington, DC 20013-7601. See 20 C.F.R. § 725.478 and § 725.479. A copy of a notice of appeal must also be served on Donald S. Shire, Esquire, Associate Solicitor for Black Lung Benefits. His address is Frances Perkins Building, Room N-2117, 200 Constitution Avenue, NW, Washington, DC 20210.

Attachment No. 1

American Board of Medical Specialties«

Certification:

Larry W. Joyce, MD
Richlands, Virginia

Certified by: The American Board of Pathology:

Anatomic and Clinical Pathology

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